EXHIBIT 1

PATENT: OC01617K

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re Application of:	Examiner: Dr. T. C. McKenzie
T. Guzi *et al.*	Group Art Unit: 1624
Serial No.: **10/654,546**	
Filed: **September 3, 2003**	Atty. Docket No.: OC01617K
For: **"Novel Pyrazolopyrimidines as Cyclin: Dependent Kinase Inhibitors"**	

Commissioner for Patents
P. O. Box 1450
Alexandria, VA 22313-1450

Sir:

DECLARATION UNDER 37 C.F.R. § 1.132

I, Timothy J. Guzi, Ph.D. declare and state as follows:

1. I received the degree Bachelor of Arts in Chemistry from St. Lawrence University in 1989, and then the degree Doctor of Philosophy in Organic Chemistry from the University of Virginia in 1994.

2. From 1994-1996, I conducted post-doctoral research in the field of synthetic organic chemistry at the Rensselaer Polytechnic Institute. I have been employed by Schering-Plough Research Institute in the laboratories located at Kenilworth, New Jersey since 1996, performing research to develop and study new molecules for cancer therapy. My present title is Associate Director and I direct a project team concerned with developing new kinase inhibitors.

3. I am named as an inventor in the subject patent application.

4. My work has included preparing and characterizing new molecules as cyclin dependent kinase inhibitors. I developed several compounds for such utility which are disclosed and claimed in the subject patent application. The assay of the compounds for cyclin dependent kinase2

(CDK2) activity is also disclosed therein, along with the IC_{50} data for the CDK2 activity for the compounds.

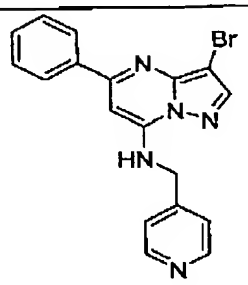
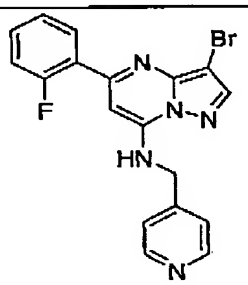
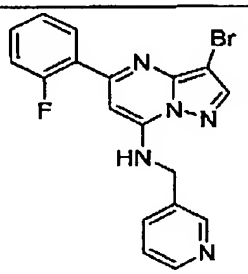
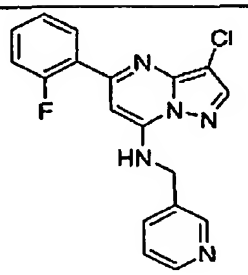
5. In addition to CDK2 activity, the compounds were also tested for cyclin dependent kinase1 (CDK1) activity. The assay employed and the activity data are reported below.

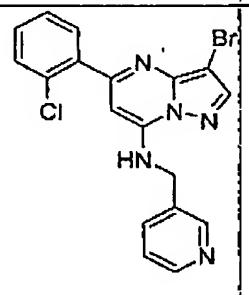
6. IN VITRO KINASE ASSAY: CDK1 kinase assay (cyclin B) was performed in low protein binding 96-well plates (Corning Inc, Corning, New York). Enzyme was diluted to a final concentration of 1.1 μg/ml in kinase buffer containing 50mM Tris pH 8.0, 10mM $MgCl_2$, 1mM DTT, and 0.1mM sodium orthovanadate. The substrate used in these reactions was a biotinylated peptide derived from Histone H1 (from Amersham, UK). The substrate was thawed on ice and diluted to 2.5 uM in kinase buffer. Compounds were diluted in 10%DMSO to desirable concentrations. For each kinase reaction, 20 μl of the 1.1 μg/ml enzyme solution (0.022 μg of enzyme) and 20 μl of the 2.5 μM substrate solution were mixed, then combined with 10 μl of diluted compound in each well for testing. The kinase reaction was started by addition of 50 μl of 2 μM ATP and 0.25 μCi of 33P-ATP (from Amersham, UK). The reaction was allowed to run for 45 min at room temperature. The reaction was stopped by adding 100 ul of stop buffer containing 2 M NaCl with 1% phosphoric acid , and 5 mg/ml streptavidine coated SPA beads (from Amersham, UK) for 15 minutes. The SPA beads were then captured onto a 96-well GF/B filter plate (Packard/Perkin Elmer Life Sciences) using a Filtermate universal harvester (Packard/Perkin Elmer Life Sciences.). Non-specific signals were eliminated by washing the beads twice with 2M NaCl then twice with 2 M NaCl with 1% phosphoric acid. The radioactive signal was then measured using a TopCount 96 well liquid scintillation counter (from Packard/Perkin Elmer Life Sciences).

IC_{50} DETERMINATION: Dose-response curves were plotted from inhibition data generated, each in duplicate, from 8 point serial dilutions of inhibitory compounds. Concentration of compound was plotted against % kinase activity, calculated by CPM of treated samples divided by CPM of untreated samples. To generate IC_{50} values, the dose-response curves were

then fitted to a standard sigmoidal curve and IC_{50} values were derived by nonlinear regression analysis. The thus-obtained IC_{50} values for representative compounds of the invention are shown in **Table A.**

Table A

CMPD	CDK1 IC_{50} (μM)
	0.03
	0.07
	0.035
	0.035

	0.012

7. The data demonstrate that the compounds of the invention are potent inhibitors of CDK1, in addition to being potent inhibitors of CDK2.

I further declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under section 1001 of Title 18 of the United States Code, and that such willful false statements may jeopardize the validity of the application or any patent issued thereon.

Further declarant sayeth not.

December 15, 2005 — Date

Timothy J. Guzi